phils to leukotriene B_4. Although much is still unknown regarding the role of the leukotrienes in inflammation, their potency and ubiquity make them strong candidates as inflammatory mediators or modulators in many disease processes involving recruitment of inflammatory cells. Present efforts are being directed toward the development of specific inhibitors of leukotriene formation. Such agents would be very useful for basic research on the interaction of arachidonic acid metabolites and of great therapeutic potential in the treatment of asthma, rheumatoid arthritis or other related conditions.

Elucidation of increasing numbers of surface receptors for diverse chemoattractants on neutrophils, together with the understanding that the various neutrophil functions are triggered by occupation of these receptors, provides basic information for a clearer understanding of neutrophil function in inflammatory conditions and will likely provide clinicians with more disease-specific and effective therapies in the near future.

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Clinical Significance of Helper/Suppressor T Cells

THE MORPHOLOGICAL determination (typing) of the percent and absolute number of circulating T-"helper" and -"suppressor" lymphocytes has gained widespread popularity with the development of monoclonal antibodies to detect antigens on the surface of different subpopulations of human lymphocytes. In this context, the terms T "helper" and "suppressor," which are functional terms, are gross oversimplifications. The helper T cells, as detected by the monoclonal antibodies in general use such as T4, contain not only helper cells for immunoglobulin synthesis but also inducer cells for many T-cell functions and even the precursors of some suppressor T cells. Thus, the T-helper/inducer population is really a mixture of related but distinct T cells. These distinctions based on function can be delineated immunologically by other monoclonal antibodies not in general use. Similarly, the suppressor T cells defined by monoclonal antibodies such as T8 comprise a variety of cell types involved in cytotoxicity and in inhibition of immune responses.

The measurement of helper and suppressor T-cell numbers, percents and ratios can be compared with the measurement of different types of leukocytes by differential cell count. Values may be abnormal in many cases but the clinical utility is limited to a very small number of disorders. Furthermore, while the test is very sensitive, it is also nonspecific, being altered in many infectious (especially viral), metabolic, neoplastic, rheumatologic and congenital and acquired immunologic disorders. Quantitation of helper/suppressor T cells may be clinically useful in certain suspected immunologic disorders. In infants, it can be used to distinguish between transient hypogammaglobulinemia (reduced helper T cells) and congenital agammaglobulinemia (normal helper/suppressor T cells and absent B cells). In adults, T-cell subsets are most often measured in the evaluation of persons suspected of having the acquired immune deficiency syndrome (AIDS). While a reduced number (and percent) of helper T cells with a normal or increased percent of suppressor cells is almost uniformly found in patients who have AIDS, these findings are very nonspecific and, in themselves, not diagnostic. Moreover, altered helper/suppressor T-cell ratios are found after a viral infection and in many sexually active homosexual men who do not have AIDS. Thus, while an abnormal helper/suppressor T-cell ratio is a characteristic immunologic finding useful in confirming the diagnosis of AIDS, it is by no means diagnostic of the disorder. In other adult immunodeficiencies, such as common variable immunodeficiency, T-cell subsets (by the generally used markers) are often abnormal but appear to have no relationship to the in vivo or in vitro functional defects observed.

Measurement of helper/suppressor T cells is not a screening test. In selected patients with possibly abnormal T-cell function, it should be used in conjunction with other tests of immune function such as a battery of delayed hypersensitivity skin tests. It is important to emphasize that patients should not be labelled as having an "immune disorder" or "dysfunction" solely on the basis of an in vitro morphologic determination of helper/suppressor T cells.

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Penicillin-Induced Anaphylaxis

OF AN ESTIMATED 400 to 800 anaphylactic deaths per year in the United States, as many as 75% have been ascribed to penicillin sensitivity. Anaphylaxis may occur in one to four instances per 10,000 patient treatment courses. The death rate is about 1 to 2 per 100,000 patients treated. The oral route appears to be the safest but reactions and even death have occurred from penicillin taken orally. Children are at lower risk than adults.

Penicillin is the only drug whose allergenic metabolites have been identified. The penicilloyl moiety, formed in largest quantity and thus called the major determinant, is responsible most often for accelerated urticarial reactions. A skin test reagent for detecting sensitivity to this determinant is available commercially as penicilloyl-poly-